********** STN Columbus **********
FILE 'HOME' ENTERED AT 13:46:34 ON 12 OCT 1999
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=> e petersen jacob s/au

E1 2 PETERSEN JACKIE Y/AU
E2 6 PETERSEN JACOB/AU
E3 28 --> PETERSEN JACOB S/AU
E4 1 PETERSEN JACOB STEEN/AU
E5 9 PETERSEN JACOB STEN/AU
E6 1 PETERSEN JACQUE/AU
E7 2 PETERSEN JAMES/AU
E8 1 PETERSEN JAMES A/AU
E9 1 PETERSEN JAMES B/AU
E10 1 PETERSEN JAMES C/AU
E11 8 PETERSEN JAMES H/AU
E12 6 PETERSEN JAMES J/AU

=> s e3-e5

L1 38 ("PETERSEN JACOB S"/AU OR "PETERSEN JACOB STEEN"/AU OR "PETERSEN JACOB STEN"/AU)

=> dup rem l1

PROCESSING COMPLETED FOR L1
L2 28 DUP REM L1 (10 DUPLICATES REMOVED)

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 28 ANSWERS - CONTINUE? Y/(N):y

L2 ANSWER 1 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1999:371016 BIOSIS
DN PREV199900371016
TI Screening for GAD antibodies in the general population-Influence of age and neuroendocrine conditions on threshold definition and false positivity.
AU Batstra, Manou R. (1); Driel, Arianne (1); ***Petersen, Jacob S. (1)*** ; Drenth, Patricia P. (1); Donselaar, Cees A. (1); Tol, Maarten J. (1); Bruining, G. Jan (1); Grobbee, Diederick E. (1); Dyrberg, Thomas (1); Aanstoot, Henk-Jan (1)
CS (1) Rotterdam Netherlands
SO Diabetes, (1999) Vol. 48, No. SUPPL. 1, pp. A432-A433.
Meeting Info.: 59th Scientific Sessions of the American Diabetes Association San Diego, California, USA June 19-22, 1999 American Diabetes Association
. ISSN: 0012-1797.
DT Conference
LA English

L2 ANSWER 2 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1998:708957 CAPLUS
DN 129:335780

TI Combinations of antigen and mucosal binding component for inducing specific immunological tolerance
IN ***Petersen, Jacob Sten***
PA Zymogenetics, Inc., USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9847529	A1	19981029	WO 1998-US8361	19980423
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9871610	A1	19981113	AU 1998-71610	19980423

PRAI US 1997-44182 19970423
US 1997-44184 19970423
WO 1998-US8361 19980423

AB This invention provides combinations of a tolerance-inducing antigen such as insulin and a mucosal binding component that preferably binds ganglioside GM1. The components are present in a non-covalent arrangement. When administered to a mucosal surface, the combinations are effective in inducing specific immunol. tolerance at a 10-fold lower dose than antigen alone. Tolerance is sustained for a no. of weeks without the necessity of booster administrations. The compns. and procedures of this invention are of benefit for the prevention or amelioration of conditions attributable to an unwanted immunol. response.

L2 ANSWER 3 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1998:480445 BIOSIS
DN PREV199800480445
TI Immunization of diabetes-prone or non-diabetes-prone mice with GAD65 does not induce diabetes or islet cell pathology.
AU Plesner, Annette (1); Worsaae, Anne; Dyrberg, Thomas; Gotfredsen, Carsten; Michelsen, Birgitte K.; ***Petersen, Jacob S.***
CS (1) Dep. Med., Box 357710, 1959 Pacific Ave., Univ. Washington, Seattle, WA 98195-7710 USA
SO Journal of Autoimmunity, (Aug., 1998) Vol. 11, No. 4, pp. 335-341. ISSN: 0896-8411.
DT Article
LA English
AB Glutamic acid decarboxylase autoimmunity was investigated by immunizing female BALB/c, C57B1/6, National Marine Research Institute (NMRI) and non-obese diabetic (NOD) mice once or twice with glumatic acid decarboxylase, GAD65, bovine serum albumin, or phosphate-buffered saline in incomplete Freunds adjuvant, or not treating. Mice immunized with GAD65, showed splinic T-cell reactivity to GAD 65 in vitro assessed by cytokine secretion. However untreated NOD mice did not. NOD mice showed a vigorous IFN-gamma response after one immunization, whereas NMRI mice

showed a lower response. IL-4 and IL-10 were only detected after two immunizations with higher levels in BALB/c, NMRI and NOD mice, compared to C57B1/6 mice. High levels of GAD65 antibodies were detected in all mice immunized with GAD65, though lower levels were found in C57B1/6 mice. Histological analysis of pancreata revealed that no control mice, regardless of treatment, had mononuclear cell infiltration in the islets. In NOD mice, peri-insulitis was detected in all groups, but less so in GAD65 and bovine serum albumin (BSA) immunized animals. These data demonstrate that NOD mice respond more vigorously to immunization with GAD65 than non-diabetic mice strains. Furthermore, immunization with GAD65 is not sufficient to provoke onset of diabetes in NOD mice or induce islet cell pathology in non-diabetes prone mice.

L2 ANSWER 4 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 1
AN 1998:119716 BIOSIS
DN PREV199800119716
TI Prediction of insulin-dependent diabetes mellitus in siblings of children with diabetes. A population-based study.
AU Kulmala, Petri; Savola, Kaisa; ***Petersen, Jacob S.*** ; Vahasalo, Paula; Karjalainen, Jukka; Lopponen, Tuija; Dyrberg, Thomas; Akerblom, Hans K.; Knip, Mikael (1); Group, Childhood Diabetes In Finland Study
CS (1) Dep. Pediatrics, Univ. Oulu, Kajaanintie 50, FIN-90220 Oulu Finland
SO Journal of Clinical Investigation, (Jan. 15, 1998) Vol. 101, No. 2, pp. 327-336.
ISSN: 0021-9738.
DT Article
LA English
AB An unselected population of 755 siblings of children with insulin-dependent diabetes mellitus (IDDM) was studied to evaluate the predictive characteristics of islet cell antibodies (ICA), antibodies to the IA-2 protein (IA-2A), antibodies to the 65-kD isoform of glutamic acid decarboxylase (GADA), insulin autoantibodies (IAA), and combinations of these markers. We also evaluated whether the histochemical ICA test could be replaced by the combined detection of other markers. 32 siblings progressed to IDDM within 7.7 yr of the initial sample taken at or close to the diagnosis of the index case (median follow-up, 9.1 yr). The positive predictive values of ICA, IA-2A, GADA, and IAA were 43, 55, 42, and 29%, and their sensitivities 81, 69, 69, and 25%, respectively. In contrast to the other three antibody specificities, GADA levels were not related to the risk for IDDM. The risk for IDDM in siblings with four, three, two, one, or no antibodies was 40, 70, 25, 2, and 0.8%, respectively. Combined screening for IA-2A and GADA identified 70% of all ICA-positive siblings, and all of the ICA-positive progressors were also positive for at least one of the three other markers. The sensitivity of the combined analysis of IA-2A and GADA was 81%, and the positive predictive value was 41%. In conclusion, combined screening for IA-2A and GADA may replace the ICA assay, giving comparable sensitivity, specificity, and positive predictive value. Accurate assessment of the risk for IDDM in siblings is complicated, as not even all those with four antibody specificities contract the disease, and some with only one or no antibodies initially will progress to IDDM.

L2 ANSWER 5 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1997:310484 BIOSIS
DN PREV199799618287

TI Population based study of prevalence of islet cell autoantibodies in monozygotic and dizygotic Danish twin pairs with insulin dependent diabetes mellitus.

AU ***Petersen, Jacob S. (1)*** ; Kyvik, Kirsten O.; Bingley, Polly J.; Gale, Edwin A. M.; Green, Anders; Dyrberg, Thomas; Beck-Nielsen, Henning

CS (1) ZymoGenetics, 1201 Eastlake Ave. East, Seattle, WA 98102 USA

SO BMJ, (1997) Vol. 314, No. 7094, pp. 1575-1579.

DT Article

LA English

AB Objective: To study the comparative importance of environment and genes in the development of islet cell autoimmunity associated with insulin dependent diabetes mellitus. Design: Population based study of diabetic twins. Setting: Danish population. Subjects: 18 monozygotic and 36 dizygotic twin pairs with one or both partners having insulin dependent diabetes. Main outcome measures: Presence of islet cell antibodies, autoantibodies, and autoantibodies to glutamic acid decarboxylase (GAD65) in serum samples from twin pairs 10 years (range 0-30 years) and 9.5 years (2-30 years) after onset of disease. Results: In those with diabetes the prevalence of islet cell antibodies, insulin autoantibodies, and autoantibodies to glutamic acid decarboxylase in the 26 monozygotic twins was 38%, 85%, and 92%, respectively, and in the dizygotic twins was 57%, 70%, and 57%, respectively. In those without diabetes the proportions were 20%, 50%, and 40% in the 10 monozygotic twins and 26%, 49%, and 40% in the 35 dizygotic twins. Conclusion: There is no difference between the prevalence of islet cell autoantibodies in dizygotic and monozygotic twins without diabetes, suggesting that islet cell autoimmunity is environmentally rather than genetically determined. Furthermore, the prevalence of islet cell antibodies was higher in the non-diabetic twins than in other first degree relatives of patients with insulin dependent diabetes. This implies that the prenatal or early postnatal period during which twins are exposed to the same environment, in contrast with that experienced by first degree relatives, is of aetiological importance.

L2 ANSWER 6 OF 28 CAPLUS COPYRIGHT 1999 ACS

AN 1997:586213 CAPLUS

DN 127:247022

TI Treatment with GAD65 or BSA does not protect against diabetes in BB rats

AU ***Petersen, Jacob Sten*** ; Mackay, Peter; Plesner, Annette; Karlsen, Allan; Gotfredsen, Carsten; Verland, Sten; Michelsen, Birgitte; Dyrberg, Thomas

CS Hagedorn Research Institute, Gentofte, DK-2820, Den.

SO Autoimmunity (1997), 25(3), 129-138
CODEN: AUIMEI; ISSN: 0891-6934

PB Harwood

DT Journal

LA English

AB The Mr 65,000 isoform of glutamic acid decarboxylase (GAD65) has been implicated as the initiating islet cell antigen in the pathogenesis of diabetes, primarily based on studies in non-obese diabetic (NOD) mice. To test the role of this islet cell autoantigen in the pathogenesis of spontaneously occurring diabetes in another animal model, purified recombinant human islet GAD65 was injected i.v. at 200 .mu.g/animal into 18-day-old diabetes-prone BB rats. For controls, bovine serum albumin (BSA), which has also been implicated in the pathogenesis of diabetes, or buffer alone was injected into age matched BB rats. At 210 days of age

there were no differences in diabetes incidence in the 3 groups, i.e. 73% (11 of 15) in the GAD65-treated, 81% (13 of 16) in the BSA-treated and 65% (11 of 17) in the buffer-treated animals, or in the median age at onset of disease, i.e. 79 days (range 65-111), 87 days (range 60-107) and 86 days (range 74-109), resp. The lack of protection against diabetes following GAD65 treatment could hypothetically be explained by no or by an aberrant expression of GAD in BB-rat islet cells. However, immunohistochem. of pancreata and immunoblotting anal. of isolated islets showed that the expression of GAD65 and GAD67 was similar in BB and Lewis rats. In conclusion, these data indicate that neither GAD65 nor BSA autoimmunity is important for the development of diabetes in BB rats, in contrast to the situation in NOD mice, and further emphasizes that extrapolation from only one animal model to autoimmune diabetes in general may not be appropriate.

L2 ANSWER 7 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1996:584152 CAPLUS
DN 125:213344
TI A method of diagnosing preclinical diabetes
IN Dragsbaek, Madsen Ole; Koch Michelsen, Birgitte; ***Petersen, Jacob*** *** Steen***
PA Novo Nordisk A/s, Den.
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9624059	A1	19960808	WO 1996-DK50	19960131
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE				
AU 9644828	A1	19960821	AU 1996-44828	19960131
EP 807253	A1	19971119	EP 1996-900889	19960131
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV				
JP 10513260	T2	19981215	JP 1996-523182	19960131
PRAI DK 1995-109		19950131		
WO 1996-DK50		19960131		

AB The present invention relates to a method of diagnosing preclin. insulin-dependent diabetes mellitus (IDDM), the method comprising contacting fetal antigen 1 (FA1) with T cells of a subject to be diagnosed for preclin. IDDM and detecting any T cell response to the FA1, such as response being indicative of preclin. IDDM in the subject.

L2 ANSWER 8 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 2
AN 1996:331153 BIOSIS
DN PREV199699053509
TI Strain-dependent differences in sensitivity of rat beta-cells to interleukin 1-beta in vitro and in vivo. Association with islet nitric oxide synthesis.
AU Reimers, Jesper I.; Andersen, Henrik Y.; Mauricio, Didac; Pociot,

Flemming; Karlsen, Allan E.; ***Petersen, Jacob S.*** ;
Mandrup-Poulsen, Thomas; Nerup, Jorn (1)
CS (1) Steno Diabetes Cent., Niels Steensenvej 2, DK-2820 Gentofte Denmark
SO Diabetes, (1996) Vol. 45, No. 6, pp. 771-778.
ISSN: 0012-1797.
DT Article
LA English
AB The aim of this study was to investigate whether strain-dependent differences in beta-cell sensitivity to interleukin (IL) 1-beta exist in vitro and in vivo and if so, whether these differences correlate to variations in IL-1-beta-induced islet inducible nitric oxide synthase (iNOS) mRNA expression and nitrite production in vitro and islet iNOS protein content in vivo. Isolated islets of Langerhans in vitro from Wistar-Kyoto/Mollegarden (WK/Mol) rats were sensitive to the inhibitory effect of IL-1-beta on accumulated and acute insulin secretion, whereas islets from Brown Norway/Charles River (BN/CR) rats were resistant. Furthermore, IL-1-beta induced higher islet iNOS mRNA expression and nitric oxide production from WK/Mol islets compared with BN/CR islets. WK/Mol, WK/CR, BN/Mol, BN/CR, and Lewis-Scripps/Mol (LS/Mol) rats received one daily injection of recombinant human IL-1-beta (4.0 mu-g/kg) or vehicle for 5 days. All the strains investigated were susceptible to IL-1-beta-induced changes in body weight, food intake, temperature, and plasma glucagon and corticosterone. However, IL-1-beta induced hyperglycemia and impairment of beta-cell glucose responsiveness in WK/Mol and LS/Mol rats, but not in BN rats. Furthermore, IL-1-beta-induced islet iNOS expression in vivo determined by immunostaining was greater in WK/Mol rats compared with WK/CR and BN/CR rats. No restriction fragment length polymorphisms, using 20 restriction enzymes, were identified in the iNOS gene in six rat strains including BioBreeding rats. In conclusion, the relative resistance of BN rat islets to IL-1-beta-induced inhibition of beta-cell function in vitro was associated with lower islet iNOS mRNA expression and nitrite production in this strain. Further, the resistance of BN rats to IL-1-beta-induced hyperglycemia was associated with a lower islet iNOS expression in vivo.

L2 ANSWER 9 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1996:93425 BIOSIS
DN PREV199698665560
TI Significance of GAD65 autoantibodies in IDDM.
AU ***Petersen, Jacob Sten (1)*** ; Buschard, Karsten; Kuhl, Claus; Molsted-Pedersen, Lars; Dyrberg, Thomas
CS (1) Hagedorn Res. Inst., Niels Steensens Vej 6, DK-2820 Gentofte Denmark
SO Baba, S. [Editor]; Kaneko, T. [Editor]. International Congress Series, (1995) No. 1100, pp. 1051-1056. International Congress Series; Diabetes, 1994.
Publisher: Elsevier Science Publishers B.V. PO Box 211, Sara Burgerhartstraat 25, 1000 AE Amsterdam, Netherlands.
Meeting Info.: 15th International Diabetes Federation Congress Kobe, Japan November 6-11, 1994
ISSN: 0531-5131. ISBN: 0-444-81725-5.
DT Book; Conference
LA English

L2 ANSWER 10 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 3
AN 1995:482958 BIOSIS

DN PREV199598497258
TI The homeodomain protein IPF-1/STF-1 is expressed in a subset of islet cells and promotes rat insulin 1 gene expression dependent on an intact E1 helix-loop-helix factor binding site.
AU Serup, Palle (1); Petersen, Helle V.; Pedersen, Erna E.; Edlund, Helena; Leonard, James; ***Petersen, Jacob S.*** ; Larsson, Lars-Inge; Madsen, Ole D.
CS (1) Hagedorn Res. Inst., Niels Steensensvej 6, Gentofte, DK-2820 Denmark
SO Biochemical Journal, (1995) Vol. 310, No. 3, pp. 997-1003.
ISSN: 0264-6021.
DT Article
LA English
AB The mouse homeodomain protein insulin promoter factor-1 (IPF-1) and the rat homologue somatostatin transactivating factor-1 (STF-1) are involved in early pancreatic development and have been implicated in the cell-specific regulation of insulin- and somatostatin-gene expression in mature islet beta- and delta-cells. The cell specificity of IPF-1/STF-1 expression in mature islets is, however, still unclear. Using antisera against recombinant IPF-1 and STF-1 in combination with antisera against islet hormones we find that all beta-cells in monolayers of newborn rat islet cells express STF-1, as do a fraction of the 8-cells. In adult rat and mouse pancreas we find a similar distribution. IPF-1/STF-1 expression was not detected in glucagon-producing alpha-cells. In islet cell tumour models we found that a glucagon/islet amyloid polypeptide (IAPP)-producing pluripotent rat islet cell line (NHI-6F-GLU) expresses STF-1 in all cells prior to insulin gene activation induced by in vivo culture. In contrast, a mouse alpha-cell line (alpha-TC1) exclusively expressed IPF-1 in a small subset of insulin-producing cells while an insulin-negative subclone (alpha-TC1.9) was negative for IPF-1. In transfection experiments using alpha-TC1.9 cells STF-1 activated a rat insulin 1 reporter gene dependent not only on both STF-1-binding sites, but also on the E1-binding site for the helix-loop-helix factor IEF-1. However, the endogenous mouse insulin genes remained inactive in these cells. These results suggest that the insulin promoter acquires its very high, yet cell-specific, activity at least partly through the action of IPF-1/STF-1. This action is dependent on helix-loop-helix factors bound to the E1 element.

L2 ANSWER 11 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 4
AN 1995:407400 BIOSIS
DN PREV199598421700
TI Cloning and Expression of Cytokine-Inducible Nitric Oxide Synthase cDNA From Rat Islets of Langerhans.
AU Karlsen, Allan E. (1); Andersen, Henrik U.; Vissing, Henrik; Larsen, Peter Mose; Fey, Stephen J.; Cuartero, Beatriz G.; Madsen, Ole D.; ***Petersen, Jacob S.*** ; Mortensen, Steen B.; Mandrup-Poulsen, Thomas; Boel, Esper; Nerup, Jorn
CS (1) Steno Diabetes Cent., Niels Steensensvej 2, 2820 Gentofte Denmark
SO Diabetes, (1995) Vol. 44, No. 7, pp. 753-758.
ISSN: 0012-1797.
DT Article
LA English
AB An inducible nitric oxide (NO) synthase isoform (iNOS) is specifically induced in the beta-cells of interleukin (IL)1-beta-exposed rat islets, suggesting a role for NO in the pathogenesis of type I diabetes. The aim of this study was to clone and characterize iNOS cDNA from

cytokine-exposed islets. Neither NO production nor iNOS transcription could be detected in rat islets or in rat insulinoma RIN-5AH beta-cells cultured in the absence of cytokines. Addition of IL-1-beta alone or in combination with tumor necrosis factor-alpha induced a concentration- and time-dependent expression of the iNOS gene and associated NO production (measured as nitrite) from both islets and RIN cells. iNOS transcripts were cloned by reverse transcriptase-polymerase chain reaction from the cytokine-exposed rat islets and RIN cells, and DNA sequence analysis revealed a near 100% identity to the recently published iNOS cDNA cloned from cytokine-exposed rat hepatocytes and smooth muscle cells. Recombinant rat islet iNOS was transiently and stably expressed in human kidney 293 fibroblasts, and the high enzymatic activity was inhibited by addition of the L-arginine analogs, N-omega-nitro-L-arginine methyl ester and aminoguanidine. Two-dimensional gel electrophoresis revealed the recombinant iNOS as a series of spots with the expected molecular mass of 131 kDa and pI values in the range of 6.8 to 7.0. In conclusion, the IL-1-beta-induced iNOS cloned and expressed from rat islets and RIN cells is encoded by the same transcript as the iNOS induced in other cell types.

L2 ANSWER 12 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1995:208094 BIOSIS
DN PREV199598222394
TI Heterogeneity of islet pathology in two infants with recent onset diabetes mellitus.
AU Lernmark, Ake (1); Kloppel, Gunter; Stenger, David; Vathanaprida, Chare; Falt, Kaj; Landin-Olsson, Mona; Baskin, Denis G.; Palmer, Jerry P.; Gown, Allen M.; ***Petersen, Jacob S.*** ; Li, Linsong; Edenvall, Hans; Mauseth, Richard S.
CS (1) Dep. Med., Univ. Washington, Seattle, WA 98195 USA
SO Virchows Archiv, (1995) Vol. 425, No. 6, pp. 631-640.
ISSN: 0945-6317.
DT Article
LA English
AB The mechanisms by which the beta cells of pancreatic islets are destroyed in insulin-dependent diabetes mellitus (IDDM) are poorly understood. In this report the pancreatic histo- and immunopathology of two children, both HLA-DR 3/4, DQ 2/8 positive and who both died from cerebral oedema within a day of clinical diagnosis of IDDM, were investigated. Patient 1, a 14-month-old girl, had a 4-week history of polydipsia and polyuria. Patient 2, a 3-year-old boy, had 2 days of illness. Both patients had a similarly severe loss of insulin cells but differed markedly as to the extent of lymphocytic islet infiltration (insulitis). Apart from insulitis, marked islet macrophage infiltration was demonstrated in both patients with the HAM-56 monoclonal antibody. Neither patient showed aberrant expression of HLA class II antigens on insulin-immunoreactive cells, but allele-specific HLA-DQ8 expression was evident on endothelial cells. Glutamic acid decarboxylase immunoreactivity was detected in both insulin- and glucagon-immunoreactive cells. It is concluded that the heterogeneity of islet pathology, especially insulitis, may reflect different dynamics and extent rather than different pathomechanisms of immune destruction of islets in IDDM.

L2 ANSWER 13 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1995:639575 CAPLUS
DN 123:223686

TI Islet expression of Rhombotin and Isl-1 suggests cell type specific exposure of LIM-domain epitopes
AU Lund, Kaare; ***Petersen, Jacob S.*** ; Jensen, Jan; Blume, Niels; Edlund, Thomas; Thor, Stefan; Madsen, Ole D.
CS Hagedorn Res. Inst., Gentofte, DK2820, Den.
SO Endocrine (1995), 3(6), 399-408
CODEN: EOCRE5; ISSN: 1355-008x
DT Journal
LA English
AB The homeodomain protein Isl-1 and the proto-oncogene Rhombotin (a LIM-only protein), share a double zinc-binding LIM domain and have both been implicated in neural and possibly endocrine development. Isl-1 is expressed in all endocrine cell types of the islet of Langerhans while Rhombotin mRNA expression was reported in rat insulinoma cells. We have cloned and sequenced Rhombotin cDNA from rat insulinoma (99.4% identical to human and mouse sequences) ad demonstrate that it is expressed in normal islets, intestinal tissue, and testis, in addn. to the brain; but absent in all other organs tested. Rhombotin mRNA is expressed in phenotypically distinct islet tumors (.alpha.-, .beta., and .delta.-tumors) at levels comparable to that of normal islets. Antisera raised against two distinct epitopes contained within a short synthetic peptide representing part of the N-terminal LIM domain of Rhombotin surprisingly stain .alpha.- and .delta.-cells, resp., on sections of rat pancreas. Rhombotin is undetectable by immunocytochem. using LIM-domain antisera on intact monolayer islet tumor cells or transfected fibroblasts while readily detectable when equipped with a FLAG epitope, as detected with FLAG antiserum. In contrast, recombinant FLAG-Rhombotin is efficiently recognized by Western blotting or immunopptn. with all LIM-specific antisera. Almost identical results were obtained with LIM-specific vs. homeodomain/C-terminal Isl-1 antisera staining .alpha.-cell cytoplasm or all islet nuclei, resp. We conclude that Rhombotin in addn. to Isl-1 is expressed in the islet of Langerhans and propose that the differential staining patterns obtained with antisera towards the LIM domains vs. flanking epitopes of both proteins reflect (1) cell-specific protein-protein interactions of these domains or, alternatively, (2) islet cell type specific expression of novel homologous LIM domain proteins.

L2 ANSWER 14 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 5
AN 1995:33171 BIOSIS
DN PREV199598047471
TI Neonatal tolerization with glutamic acid decarboxylase but not with bovine serum albumin delays the onset of diabetes in NOD mice.
AU ***Petersen, Jacob Sten (1)*** ; Karlsen, Allan E.; Markholst, Helle; Worsaae, Anne; Dyrberg, Thomas; Michelsen, Birgitte
CS (1) Hagedorn Res. Inst., Niels Steensens Vej 6, DK-2820 Gentofte Denmark
SO Diabetes, (1994) Vol. 43, No. 12, pp. 1478-1484.
ISSN: 0012-1797.
DT Article
LA English
AB To test the role of glutamic acid decarboxylase (GAD-65) or bovine serum albumin (BSA) autoimmunity in the pathogenesis of diabetes, GAD-65 or BSA was injected intraperitoneally into neonatal female NOD mice (100 mu-g/mouse of each protein). Treatment with GAD-65, but not with BSA, significantly delayed the onset of diabetes compared with control mice (P

lt 0.05). At 18 weeks, 6 of 10 control mice compared with 0 of 10 GAD-65-treated mice (P = 0.005) and 7 of 14 BSA-treated mice had developed diabetes. However, after 79 weeks, 6 of 10 of the GAD-65-treated mice were diabetic compared with 9 of 10 of the control mice and 12 of 14 of the BSA-treated mice. In GAD-65-treated mice without diabetes, insulitis was markedly reduced compared with control or BSA-treated mice (P lt 10-4). To further elucidate why GAD becomes an autoantigen, the expression in NOD mice islets was studied. Quantitative immunohistochemistry revealed that islet cell expression of GAD was increased in 5-week-old NOD mice compared with BALB/c mice (P = 0.02). With the occurrence of insulitis (9-15 weeks), the GAD expression was further increased relative to 5-week-old NOD mice (P lt 0.02). In conclusion, GAD, but not BSA, autoimmunity is important for the development of diabetes in NOD mice. Furthermore, concordant with the appearance of insulitis, the GAD expression increased in NOD mouse islets, which could possibly potentiate the beta-cell-directed autoimmunity.

L2 ANSWER 15 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1994:547874 BIOSIS
DN PREV199598007422
TI Glutamic acid decarboxylase (GAD-65) autoantibodies in prediction of beta-cell function and remission in recent-onset IDDM after cyclosporin treatment.
AU ***Petersen, Jacob Sten (1)*** ; Dyrberg, Thomas; Karlsen, Alan E.; Molvig, Jens; Michelsen, Birgitte; Nerup, Jorn; Mandrup-Poulsen, Thomas; Group, The Canadian-European Randomized Control Trial
CS (1) Hagedorn Research Inst., Niels Steensesn Vej 6, DK-2820 Gentofte Denmark
SO Diabetes, (1994) Vol. 43, No. 11, pp. 1291-1296.
ISSN: 0012-1797.
DT Article
LA English
AB We have investigated whether glutamic acid decarboxylase (GAD) autoantibodies (GAD-65 Ab) were affected by cyclosporin therapy and were related to subsequent noninsulin-requiring remission and loss of glucagon-stimulated C-peptide response in 132 recent-onset insulin-dependent diabetes mellitus (IDDM) patients treated with cyclosporin or placebo for 12 months. GAD-65 Ab were detected in a quantitative radioligand assay using as tracer recombinant, in vitro translated, human islet (35S)methionine-labeled GAD-65. GAD65 Ab were found at onset in 66% (87 of 132) of IDDM patients and in 1% (1 of 100) of healthy control subjects. The prevalence of GAD-65 Ab and median GAD-65 Ab levels did not change in serum samples taken 3, 6, 9, and 12 months after study entry in either the cyclosporin- or the placebo-treated groups. The presence or absence of GAD-65 Ab at study entry did not predict non-insulin-requiring remission in either cyclosporin- or placebo-treated patients. However, the relative (compared with 0 months) glucagon-stimulated C-peptide response was more than 30% lower in GAD-65 Ab+ patients receiving placebo at 9 and 12 months compared with the GAD-65 Ab- placebo patients (P lt 0.035). Islet cell cytoplasmic antibody (ICA) and GAD-65 Ab+ placebo-treated patients showed no significant differences in stimulated C-peptide levels compared with those who were ICA- and GAD-65 Ab+, suggesting that ICA was not independently associated with loss of beta-cell function. We conclude that GAD-65 Ab at diagnosis may predict a more rapid loss of beta-cell function, a finding of importance when

selecting individuals at risk of developing IDDM or recent-onset EDDM patients for intervention therapy.

L2 ANSWER 16 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 6
AN 1994:451481 BIOSIS
DN PREV199497464481
TI High prevalence of autoantibodies to glutamic acid decarboxylase in long-standing IDDM is not a marker of symptomatic autonomic neuropathy.
AU Zanone, Maria M.; ***Petersen, Jacob S.*** ; Peakman, Mark; Mathias, Christopher J.; Watkins, Peter J.; Dyrberg, Thomas; Vergani, Diego (1)
CS (1) Immunol. Dep., King's Coll. Sch. Med. Dent., Bessemer Rd., London SE5 9PJ UK
SO Diabetes, (1994) Vol. 43, No. 9, pp. 1146-1151.
ISSN: 0012-1797.
DT Article
LA English
AB Immune reactivity to the enzyme glutamic acid decarboxylase (GAD), a pancreatic islet autoantigen, is present at the diagnosis of insulin-dependent diabetes mellitus (IDDM). Because GAD is also highly expressed in the nervous system, we investigated the presence of autoantibodies to the isoform GAD-65 in patients with diabetic neuropathy, which is a debilitating complication of the disease. We studied 39 patients with autonomic and somatic neuropathy, 28 patients matched for age and IDDM duration, and 13 patients with a shorter duration of IDDM, all with no diabetic complications, as well as 50 recently diagnosed diabetic patients, 23 neurologic patients with idiopathic autonomic failure unrelated to IDDM, and 72 healthy subjects. An immunoprecipitation radioligand assay was used to detect anti-GAD-65 autoantibodies with in vitro transcribed and translated human islet GAD-65 as antigen. Autoantibodies to GAD-65 were present in 56% of the diabetic patients with neuropathy, 57% of the long-duration and 69% of the short-duration diabetic control subjects, 78% of the recently diagnosed patients, and 13% of the nondiabetic neuropathic patients. Among the diabetic patients with neuropathy, there was no correlation between the presence of antiGAD-65 antibodies and the presence of autoantibodies to sympathetic ganglia, vagus nerve, or adrenal medulla structures identified by immunofluorescence. Our study shows that anti-GAD-65 antibodies are present in a high proportion of patients with diabetic neuropathy but are not exclusively associated with it, rendering it unlikely that they have a role as a disease marker or that they are pathogenetic. Our findings also show that humoral autoimmunity to GAD-65 is a major feature of diabetes of long duration (up to 52 years), although the source of the persistent stimulus for this reaction and its significance remain to be established.

L2 ANSWER 17 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1994:450174 BIOSIS
DN PREV199497463174
TI Contribution of glutamate decarboxylase antibodies to the reactivity of islet cell cytoplasmic antibodies.
AU Marshall, Michael O. (1); Hoyer, Poul E.; ***Petersen, Jacob S.*** ; Hejnaes, Kim R.; Genovese, Stefano; Dyrberg, Thomas; Bottazzo, Gian Franco
CS (1) Novo Nordisk A/S, Novo Allee, 2880 Bagsvaerd Denmark
SO Journal of Autoimmunity, (1994) Vol. 7, No. 4, pp. 497-508.
ISSN: 0896-8411.
DT Article

LA English

AB The contribution of glutamate decarboxylase (Mr 65000) antibodies to the reactivity of islet cell cytoplasmic antibodies with the 'whole' islet staining pattern from patients with newly diagnosed Type I diabetes was investigated. Diluted sera (n=10) were preincubated with increasing concentrations of purified recombinant human islet glutamate decarboxylase (Mr 65000) and the change in islet cell cytoplasmic antibody binding was evaluated by quantitative immunocytochemistry. Binding to islet cells was partially blocked by glutamate decarboxylase in 9/10 diluted sera; the maximum blocking obtained at high concentrations of glutamate decarboxylase (5 mu-g/ml) was 36% (median, range 24-61%). In contrast, binding to islet cells in three diluted sera (two polyendocrine patients without Type I diabetes and one patient with newly diagnosed Type I diabetes) with the 'selective' islet staining pattern was totally blocked by glutamate decarboxylase. The concentration of glutamate decarboxylase required to achieve maximum blocking was less for the 'whole' islet (0.4-8.0 mu-g/mu-l undiluted serum) compared to the 'selective' islet (20-645 mu-g/mu-l undiluted serum) positive sera. All sera were positive for glutamate decarboxylase antibodies in an immunoprecipitation assay using 35S-methionine labelled extract of baby hamster kidney cells transfected with glutamate decarboxylase. However, the binding activity of these antibodies was less in the sera positive for the 'whole' islet compared to the 'selective' islet staining pattern. In conclusion, glutamate decarboxylase antibodies contribute partially to the reactivity of islet cell cytoplasmic antibodies of the 'whole' islet staining pattern in the sera of newly diagnosed patients with Type I diabetes, and totally to the reactivity of the 'selective' islet staining pattern. The antigens recognized by the other antibodies contributing to the 'whole' islet reactivity remain to be defined.

L2 ANSWER 18 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS

AN 1994:160708 BIOSIS

DN PREV199497173708

TI Detection of GAD-65 antibodies in diabetes and other autoimmune diseases using a simple radioligand assay.

AU ***Petersen, Jacob S.*** ; Hejnaes, Kim R.; Moody, Alister; Karlsen, Allan E.; Marshall, Michael O.; Hoier-Madsen, Mimi; Boel, Esper; Michelsen, Birgitte K.; Dyrberg, Thomas (1)

CS (1) Novo-Nordisk A/S, novo Alle, DK-2880 Bagsvaerd Denmark

SO Diabetes, (1994) Vol. 43, No. 3, pp. 459-467.
ISSN: 0012-1797.

DT Article

LA English

AB Autoantibodies to glutamic acid decarboxylase (GAD) are frequent at or before the onset of insulin-dependent diabetes mellitus (IDDM). We have developed a simple, reproducible, and quantitative immunoprecipitation radioligand assay using as antigen in vitro transcribed and translated (35S)methionine-labeled human islet GAD-65. By using this assay, 77% (77 of 100) of serum samples from recent-onset IDDM patients were positive for GAD-65 antibodies compared with 4% (4 of 100) of serum samples from healthy control subjects. In competition analysis with unlabeled purified recombinant human islet GAD-65 binding to tracer was inhibited in 74% (74 of 100) of the GAD-65-positive IDDM serum samples compared with 2% of the control samples. The levels of GAD antibodies expressed as an index value relative to a standard serum, analyzed with or without competition, were

almost identical (r = 0.991). The intra- and interassay variations of a positive control serum sample were 2.9 and 7.6%, respectively (n = 4). The frequency of GAD antibodies was significantly higher with IDDM onset before the age of 30 (80%, 59 of 74) than after the age of 30 (48%, 10 of 21) (P lt 0.01). The prevalence of islet cell antibodies showed a similar pattern relative to age at onset. Because simultaneous occurrences of multiple autoimmune phenomena are common, we analyzed sera from patients with other autoimmune diseases. The frequency of GAD antibodies in sera positive for DNA autoantibodies (8% (2 of 25) and 4% (1 of 25) in competition analysis) or rheuma factor autoantibodies (12% (4 of 35) and 3% (1 of 35) in competition analysis) was not different from that in control samples. In contrast, in sera positive for ribonucleoprotein antibodies the frequency of GAD antibodies was significantly increased (73% (51 of 70) and 10% (7 of 70) in competition analysis (P lt 0.025)). In conclusion, even large numbers of serum samples can now be tested for GAD-65 antibodies in a relatively short time, allowing screening of individuals without a family history of IDDM for the presence of this marker.

L2 ANSWER 19 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 7
AN 1993:341937 BIOSIS
DN PREV199396038937
TI Regulation of glutamic acid decarboxylase diabetes autoantigen expression in highly purified isolated islets from Macaca nemestrina.
AU Hagopian, William A. (1); Karlsen, Allan E.; ***Petersen, Jacob S.*** ; Teague, Jeanette; Gervassi, Ana; Jiang, Jianjie; Fujimoto, Wilfred; Lernmark, Ake
CS (1) R. H. Williams Lab., Dep. Med., RG-20, Univ. Washington, Seattle, WA 98195
SO Endocrinology, (1993) Vol. 132, No. 6, pp. 2674-2681. ISSN: 0013-7227.
DT Article
LA English
AB Macaca nemestrina, which may have larger and more numerous pancreatic islets than other species, was used for large scale islet isolation by ductal collagenase perfusion and Ficoll gradient centrifugation. The average yield was 51,000 islet equivalents per pancreas, or 8,750 islets equivalents per g. The average purity was 91%, often exceeding 95%. These are the highest reported size, purity, and yield per g of any nonautomated primate islet series. Perifusion with glucose, arginine, and isobutylmethylxanthine showed appropriate biphasic insulin secretion. Unlike that in the rat, human islet glutamic acid decarboxylase (GAD) isoform expression is restricted. However, glycemic regulation of GAD expression has been shown only in rats. We, therefore, tested hypotheses that M. nemestrina islets also have restricted GAD expression, that GAD expression in primates is stimulated by glucose, and that this stimulation remains restricted to the 64,000 mol wt (GAD65) isoform. Immunoprecipitation of labeled islet extracts showed that GAD65 expression increased 16.7 +- 0.6-fold during high glucose in vitro culture. After controlling for observed increases in protein synthesis, specific glucose stimulation was still 4.2 +- 0.2-fold. Specific antisera revealed no GAD67 expression under basal conditions, and isoform restriction was maintained during stimulation. Increased GAD65 synthesis thus accounts for glucose stimulation of 64K expression. These time- and concentration-dependent effects of glucose suggest that hyperglycemia increases autoantigenicity

and may accelerate beta-cell destruction in primates, supporting a role for beta-cell rest in insulin-dependent diabetes mellitus prevention.

L2 ANSWER 20 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 8
AN 1993:227566 BIOSIS
DN PREV199395118741
TI Differential expression of glutamic acid decarboxylase in rat and human islets.
AU ***Petersen, Jacob S. (1)*** ; Russle, Steven; Marshall, Michael O.; Kofod, Hans; Buschard, Karsten; Cambon, Natalie; Karlsen, Allan E.; Boel, Esper; Hagopian, William A.; et al.
CS (1) Hagedorn Res. Inst., Niels Steensens Vej 6, DK-2820 Gentofte Denmark
SO Diabetes, (1993) Vol. 42, No. 3, pp. 484-495.
ISSN: 0012-1797.
DT Article
LA English
AB The GABA synthesizing enzyme GAD is a prominent islet cell autoantigen in type I diabetes. The two forms of GAD (GAD-64 and GAD-67) are encoded by different genes in both rats and humans. By in situ hybridization analysis of rat and human pancreases, expression of both genes was detected in rat islets, whereas only GAD-64 mRNA was detected in human islets. Immunocytochemical analysis of rat and human pancreatic sections or isolated islets with antibodies to GAD-64 and GAD-67 in combination with antibodies to insulin, glucagon, or SRIF confirmed that a GAD-64 and GAD-67 expression were beta-cell specific in rat islets. In contrast, only GAD-64 was detected in human islets and was, in addition to beta-cells, also surprisingly localized to some alpha-cells, delta-cells, and PP-cells. In long-term (4 wk) monolayer cultures of newborn rat islet cells, GAD-64 expression remained beta-cell specific as observed in vivo, whereas GAD-67 was localized not only to the beta-cells but also in the alpha-cells and delta-cells. A small but distinct fraction of GAD positive cells in these monolayer cultures did not accumulate GABA immunoreactivity, which may indicate cellular heterogeneity with respect to GABA catabolism or GAD enzyme activity. In a rat insulinoma cell line (NHI-6F) producing both glucagon and insulin depending on the culture conditions, GAD-64 expression was detected only in cultures in which the insulin producing phenotype dominated. In conclusion, these data demonstrate that the two GAD isoforms are differentially expressed in rat and human islets but also that the expression differs according to culture conditions. These findings emphasize the need to consider both the species and culture conditions of islets.

L2 ANSWER 21 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 9
AN 1993:185194 BIOSIS
DN PREV199395095644
TI Quantitative assay using recombinant human islet glutamic acid decarboxylase (GAD65) shows that 64K autoantibody positivity at onset predicts diabetes type.
AU Hagopian, William A. (1); Karlsen, Allan E.; Gottsater, Anders; Landin-Olsson, Mona; Grubin, Catherine E.; Sundkvist, Goran; ***Petersen, Jacob S.*** ; Boel, Esper; Dyrberg, Thomas; Lernmark, Ake
CS (1) R. H. Williams Lab., Dep. Med., RG-20, Univ. Wash., Seattle, WA 98195 USA
SO Journal of Clinical Investigation, (1993) Vol. 91, No. 1, pp. 368-374.
ISSN: 0021-9738.

DT Article
LA English
AB At and before onset, most insulin-dependent diabetics (IDDM) have islet GAD65 autoantibodies (GAD65Ab). Since IDDM also occurs in older patients where non-insulin-dependent diabetes is common, we studied GAD65Ab at onset to classify diabetes type. Our quantitative immunoprecipitation assay uses recombinant human islet GAD65 stably expressed in hamster fibroblasts. Electrophoretic mobility was identical to native islet GAD65. Like nativer antigen, recombinant GAD65 migrated as two bands during electrophoresis, but converted to one under stronger reduction. Immunoprecipitation was linear with respect to antibody or antigen concentration. In 120 population-based diabetic patients of all ages grouped by treatment at onset and after 18 mo, GAD65Ab were present in 70% on insulin (n = 37), 10% on oral agent (n = 62, P lt 0.0001), 69% changing from oral agent to insulin (n = 16, P lt 0.001), and 1 of 33 controls. 65% with GAD65Ab, versus 8% without, changed from oral agent to insulin (P lt 0.01). The GAD65Ab quantitative index was remarkably stable, and only 2 of 32 patients changed antibody status during follow-up. Concordance between GAD65Ab and islet cell antibodies was 93%. Quantitative correlation was approximate but significant. This highly sensitive, quantitative, high capacity assay for GAD65Ab reveals treatment requirements better than clinical criteria, perhaps guiding immunomodulatory therapy.

L2 ANSWER 22 OF 28 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 10
AN 1993:242868 BIOSIS
DN PREV199344116068
TI Differential islet cell expression of two glutamate decarboxylases, both autoantigens in diabetes.
AU Michelsen, Birgitte Koch (1); ***Petersen, Jacob S.*** ; Rambrandt, Tina B.; Boel, Esper; Karlsen, Allan E.; Videbaek, Nicoline; Blume, Niels; Madsen, Ole D.
CS (1) Hagedorn Research Inst., Gentofte Denmark
SO Biochemical Society Transactions, (1993) Vol. 21, No. 1, pp. 173-177.
Meeting Info.: 644th Meeting of the Biochemical Society Glasgow, Scotland, UK September 16-18, 1992
ISSN: 0300-5127.
DT Article
LA English

L2 ANSWER 23 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1993:5493 CAPLUS
DN 118:5493
TI Recombinant glutamic acid decarboxylase (representing the single isoform expressed in human islets) detects IDDM-associated 64,000-Mr autoantibodies
AU Karlsen, Allan E.; Hagopian, William A.; ***Petersen, Jacob S.*** ; Boel, Esper; Dyrberg, Thomas; Grubin, Catherine E.; Michelsen, Birgitte K.; Madsen, Ole D.; Lernmark, Aake
CS Dep. Endocrinol., Karolinska Hosp., Stockholm, Swed.
SO Diabetes (1992), 41(10), 1355-9
CODEN: DIAEAZ; ISSN: 0012-1797
DT Journal
LA English
AB Glutamic acid decarboxylase (GAD) is an autoantigen in insulin-dependent diabetes mellitus (IDDM). Mol. cloning and specific antibodies

demonstrate that only the lower mol. wt. GAD64 isoform is expressed in human islets, in contrast to human brain, rat islets, and rat brain, all of which express both GAD64 and GAD67. Expression of the human islet GAD64 isoform in COS-7 and BHK cells resulted in an enzymically active rGAD64, which is immunoreactive with diabetic sera comparable with that of the islet 64,000-mol. wt. autoantigen. Immunopptn. analyses showed that 21/28 (75%) IDDM sera had rGAD64 antibodies compared with only 1/59 (1.7%) of the healthy control sera. In immunoblot analyses, a stiff-man syndrome serum, but only 1/10 randomly selected IDDM sera recognized the blotted rGAD64 without relation to immunopptn. titers. Thus, only the GAD64 isoform is expressed in human islets, in contrast to rat islets, which also express the GAD67 isoform. The immunol. properties of human rGAD64 are comparable with the native 64,000-mol. wt. islet autoantigen, allowing further studies of the immunopathogenesis of IDDM.

L2 ANSWER 24 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1992:208177 CAPLUS
DN 116:208177
TI Immature transformed rat islet .beta.-cells differentially express C-peptides derived from the genes coding for insulin I and II as well as a transfected human insulin gene
AU Blume, Niels; ***Petersen, Jacob S.*** ; Andersen, L. CHristina; Kofod, Hans; Dyrberg, Thomas; Michelsen, Birgitte K.; Serup, Palle; Madsen, Ole D.
CS Hagedorn Res. Lab., Gentofte, DK 2820, Den.
SO Mol. Endocrinol. (1992), 6(2), 299-307
CODEN: MOENEN; ISSN: 0888-8809
DT Journal
LA English
AB Synthetic peptides representing unique sequences in rat proinsulin C-peptide I and II were used to generate highly specific antisera, which, when applied on sections of normal rat pancreas, confirm a homogeneous coexpression of the two C-peptides in all islet .beta.-cells. Insulin gene expression is induced in the transformed heterogeneous rat islet cell clone, NHI-6F, by transient in vivo passage. During this process a transfected human insulin gene is coactivated with the endogenous nonallelic rat insulin I and II genes. Newly established cultures from NHI-6F insulinomas having a high frequency of insulin-producing cells showed highly differential expression at the cellular level of the three proinsulin C-peptide immunoreactivities, as follows: C-peptide I > human C-peptide > C-peptide II. The fractions of cells expressing human C-peptide and C-peptide I-producing population was still present. Double-labeling expts. revealed a heterogeneous distribution of the three different C-peptides. Surprisingly, in the early passages a large fraction of cells would express only a single species of proinsulin-C peptide immunoreactivity but still at high levels. However, rat C-peptide II and human C-peptide were often colocalized, even in later passages. In situ hybridization studies combined with the immunocytochem. data suggest that the differential expression occurs at the level of transcription. Thus, NHI-6F cells may represent a level of .beta.-cell differentiation characterized by a phenotype in which only a single insulin gene is active and that the insulin I gene is preferentially expressed under these conditions. The common behavior, i.e. coexpression and gradual inactivation during successive culture, of a transfected human insulin gene with the homologous rat insulin II gene may suggest the presence of

common regulatory characteristics not shared with the retroposed rat insulin I gene.

L2 ANSWER 25 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1993:20612 CAPLUS
DN 118:20612
TI Production of epitope specific monoclonal IgG antibodies to HLA class II molecules by combining in vivo and in vitro immunization
AU ***Petersen, Jacob Sten*** ; Dyrberg, Thomas
CS Hagedorn Res. Lab., Gentofte, DK-2820, Den.
SO J. Immunol. Methods (1992), 151(1-2), 15-26
CODEN: JIMMBG; ISSN: 0022-1759
DT Journal
LA English
AB To study the expression of HLA-DQ .beta. chain alleles assocd. with type I diabetes, monoclonal antibodies (mAbs) were generated from mice immunized with synthetic peptides representing allelic HLA-DQw7 and HLA-DQw8 .beta. chain sequences. The splenocytes from immunized mice were fused with myeloma cells, either immediately after or following addnl. in vitro boosting with peptide. Peptide-specific mAbs, predominantly of the IgG isotype, were isolated only from in vitro boosted splenocytes. Immunoblot anal. showed that several of the mAbs cross-reacted with DQ .beta. chain mols. One mAb to a peptide representing DQw8.beta. position [49-60] specifically recognized the DQ28 .beta. chain. Three mAbs to a peptide representing DQw8.beta. position [39-52] specifically recognized an epitope consisting of Gly-Val-Tyr in position 45-47, i.e., all DQ.beta. alleles except DQw7.beta. (position 45-47: Glu-Val-Tyr) and DQw2.beta. (position 45-47; Gly-Glu-Phe). In FACS anal. these mAbs bound lymphocytes with the same specificity as found by immunoblotting anal. Thus, by combining in vivo and in vitro immunization a no. of epitope specific monoclonal IgG antibodies were generated that distinguish closely related HLA-DQ .beta. chain alleles in predetd. positions.

L2 ANSWER 26 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1992:565040 CAPLUS
DN 117:165040
TI Cloning, characterization, and autoimmune recognition of rat islet glutamic acid decarboxylase in insulin-dependent diabetes mellitus
AU Michelsen, Birgitte Koch; ***Petersen, Jacob Sten*** ; Boel, Esper; Moeldrup, Annette; Dyrberg, Thomas; Madsen, Ole Dragsbaek
CS Hagedorn Res. Lab., Gentofte, DK-2820, Den.
SO Proc. Natl. Acad. Sci. U. S. A. (1991), 88(19), 8754-8
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
AB A 64-kDa islet protein is a major autoantigen in insulin-dependent diabetes mellitus (IDDM). Antiantibodies against the 64-kDa protein was recently shown to immunoppt. glutamic acid decarboxylase (GAD, EC 4.1.1.15) from brain and from islets. Evidence is presented that the autoantisera also recognizes a hydrophilic islet protein of .apprxeq.67 kDa in addn. to the amphiphilic 64-kDa form. A full-length rat islet GAD cDNA encoding a hydrophilic 67-kDa protein, which appears to be identical to rat brain 67-kDa GAD was isolated. A partial sequence of human insulinoma 67-kDa GAD was identical to human brain 67-kDa GAD. Allelic variations were obsd. in rat as well as in human 67-kDa GAD sequences.

The expressed rat islet 67-kDa GAD protein is functional and is immunopptd. by IDDM sera; it comigrates electrophoretically with the 67-kDa islet autoantigen. The hydrophilic 67-kDa form of GAD in islets is an addnl. autoantigen in the IDDM and is recognized by a different subset of autoantibodies than the 64-kDa autoantigen. Thus, mammalian cell lines expressing functionally active, recombinant GAD may become important tools to study the nature and the role of GAD autoreactivity in IDDM.

L2 ANSWER 27 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1991:653949 CAPLUS
DN 115:253949
TI Interferon stimulates the expression of 2',5'-oligoadenylate synthetase and MHC class I antigens in insulin-producing cells
AU Bonnevie-Nielsen, Vagn; Gerdes, Anne Marie; Fleckner, Jan; ***Petersen,*** *** Jacob S.*** ; Michelsen, Birgitte; Dyrberg, Thomas
CS Dep. Clin. Chem., Odense Univ. Hosp., Odense, 5000, Den.
SO J. Interferon Res. (1991), 11(5), 255-60
CODEN: JIREDJ; ISSN: 0197-8357
DT Journal
LA English
AB The pathogenesis of type 1 diabetes involves autoimmune processes directed against the pancreatic .beta.-cells. The etiol. is not known, but circumstantial evidence suggests a connection between virus infection and development of the disease. Therefore, because the interferon-(IFN) dependent 2',5'-oligoadenylate (2-5A) synthetase system constitutes an important part of the nonspecific immune defense against viral infections, the activity of the enzyme was examd. in islets of Langerhans, RIN cells, and GH3 cells. First, the 2-5A synthetase was expressed constitutively in all cell types and, second, all cells were sensitive to stimulation with IFN-.alpha.. The 2-5A synthetase activity induced by 1000 U/mL of IFN-.alpha. increased by 400% in pancreatic islets and by more than 1000% in GH3 and RIN cells. However, the IFN-.alpha. concn. needed to induce half-maximal 2-5A synthetase activity was nearly the same in the 3 cell types (i.e., ranging from 59 to 66 U/mL IFN-.alpha.). The 2-5A synthetase present in islets and RIN cells was highly sensitive to poly (I:C). In pancreatic islets and RIN cells, the 2-5A synthetase enzyme generated dimers and trimers of 2',5'-oligoadenylates. Furthermore, exposure of RIN cells to IFN-.alpha. showed an increase in MHC class I expression already at 5 U/mL and maximal expression at about 200 U/mL IFN-.alpha.. The examd. endocrine cells express the 2-5A synthetase enzyme as well as MHC class I antigen constitutively, but also by stimulation with IFN in vitro. This part of the immune defense system, therefore, is active in the basal state and is readily activated in the presence of IFN in endocrine cells.

L2 ANSWER 28 OF 28 CAPLUS COPYRIGHT 1999 ACS
AN 1990:116896 CAPLUS
DN 112:116896
TI Immunological cross-reactivity between mimicking epitopes on a virus protein and a human autoantigen depends on a single amino acid residue
AU Dyrberg, Thomas; ***Petersen, Jacob Sten*** ; Oldstone, Michael B. A.
CS Hagedorn Res. Lab., Gentofte, Den.
SO Clin. Immunol. Immunopathol. (1990), 54(2), 290-7
CODEN: CLIIAT; ISSN: 0090-1229
DT Journal
LA English

AB A novel autoantigenic region has been identified on the .alpha. chain of the human acetylcholine receptor (HuAChR), residues 160-167. Antibodies to that sequence appeared in the sera of a subset of patients with myasthenia gravis (MG), and affinity-purified antibody was biol. active for an in vitro assay of the HuAChR. Sequence homol. between the autoantigen and 2 sep. regions of herpes simplex virus glycoprotein D (HSV-GpD) have been identified. These components are, resp. the HuAChR, residues [160-167] (PESDQPDL), and residues [286-293] (PNATQPEL) and [381-388] (PEDDQPSS) of HSV-GpD. Antisera from rabbits immunized with synthetic peptides representing HSV-GpD [286-293] bound strongly to the AChR peptide. In contrast, antiserum to HSV-GpD [381-388] bound minimally, if at all, even though both virus peptides shared 4 amino acids with the receptor sequence. To investigate the mol. basis for the differential binding, the authors tested the reactivity of the HSV-GpD antisera to analogs of the [381-388] virus peptide contg. single amino acid substitutions. The results demonstrated that the cross-reactivity between HuAChR [160-167] and HSV-GpD [286-293] predominantly depends on a single residue, the C-terminal leucine in HSV-GpD, position 293.

=> s (ctb or (cholera toxin b)) and toler?

L3 165 (CTB OR (CHOLERA TOXIN B)) AND TOLER?

=> dup rem l3

PROCESSING COMPLETED FOR L3
L4 64 DUP REM L3 (101 DUPLICATES REMOVED)

=> d 1-

YOU HAVE REQUESTED DATA FROM 64 ANSWERS - CONTINUE? Y/(N):y

L4 ANSWER 1 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 1999:625445 SCISEARCH
GA The Genuine Article (R) Number: 224AU
TI Treatment of experimental autoimmune arthritis by nasal administration of a type II collagen-cholera toxoid conjugate vaccine
AU Tarkowski A (Reprint); Sun J B; Holmdahl R; Holmgren J; Czerkinsky C
CS DEPT RHEUMATOL, GULDHEDSGATAN 10, S-41346 GOTHENBURG, SWEDEN (Reprint); UNIV GOTHENBURG, GOTHENBURG, SWEDEN; LUND UNIV, LUND, SWEDEN; FAC MED NICE, INSERM, F-06034 NICE, FRANCE
CYA SWEDEN; FRANCE
SO ARTHRITIS AND RHEUMATISM, (AUG 1999) Vol. 42, No. 8, pp. 1628-1634.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106.
ISSN: 0004-3591.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 27
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 2 OF 64 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 1

AN 1999:509105 CAPLUS
TI Suppressive versus stimulatory effects of allergen/cholera toxoid (***CTB***) conjugates depending on the nature of the allergen in a murine model of type I allergy
AU Wiedermann, Ursula; Jahn-Schmid, Beatrice; Lindblad, Marianne; Rask, Carola; Holmgren, Jan; Kraft, Dietrich; Ebner, Christof
CS Division of Immunopathology, Institute of General and Experimental Pathology, University of Vienna, Vienna, 1090, Austria
SO Int. Immunol. (1999), 11(7), 1131-1138
CODEN: INIMEN; ISSN: 0953-8178
PB Oxford University Press
DT Journal
LA English

L4 ANSWER 3 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 1999:554197 SCISEARCH
GA The Genuine Article (R) Number: 215AY
TI Intranasal administration of a Schistosoma mansoni glutathione S-transferase-cholera toxoid conjugate vaccine evokes antiparasitic and antipathological immunity in mice
AU Sun J B (Reprint); Mielcarek N; Lakew M; Grzych J M; Capron A; Holmgren J; Czerkinsky C
CS GOTHENBURG UNIV, DEPT MED MICROBIOL & IMMUNOL, GULDHEDSGATAN 10, S-41346 GOTHENBURG, SWEDEN (Reprint); INST PASTEUR, INSERM U167, CTR IMMUNOL & BIOL PARASITAIRE, F-59019 LILLE, FRANCE; UNIV ADDIS ABABA, DEPT BIOL, ADDIS ABABA, ETHIOPIA; INSERM U364, NICE, FRANCE
CYA SWEDEN; FRANCE; ETHIOPIA
SO JOURNAL OF IMMUNOLOGY, (15 JUL 1999) Vol. 163, No. 2, pp. 1045-1052.
Publisher: AMER ASSOC IMMUNOLOGISTS, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0022-1767.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 50
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 4 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 1999:141122 SCISEARCH
GA The Genuine Article (R) Number: 165MF
TI Intranasal or subcutaneous co-administration of recombinant ***cholera*** ***toxin*** ***B*** subunit stimulates only a slight or no level or the specific IgE response in mice to tetanus toxoid
AU Isaka M; Yasuda Y; Kozuka S; Taniguchi T; Miura Y; Matano K; Goto N; Tochikubo K (Reprint)
CS NAGOYA CITY UNIV, SCH MED, DEPT MICROBIOL, MIZUHO KU, NAGOYA, AICHI 4678601, JAPAN (Reprint); NAGOYA CITY UNIV, SCH MED, DEPT MICROBIOL, MIZUHO KU, NAGOYA, AICHI 4678601, JAPAN; NATL INST INFECT DIS, DEPT SAFETY RES BIOL, TOKYO 2080011, JAPAN
CYA JAPAN
SO VACCINE, (26 FEB 1999) Vol. 17, No. 7-8, pp. 944-948.
Publisher: ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND.
ISSN: 0264-410X.
DT Article; Journal

FS LIFE; AGRI
LA English
REC Reference Count: 28
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 5 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 1999154078 EMBASE
TI Transgenic plants as edible vaccines.
AU Richter L.; Kipp P.B.
CS L. Richter, Boyce Thompson Inst. Plant Res. Inc., Tower Rd., Ithaca, NY 14850, United States
SO Current Topics in Microbiology and Immunology, (1999) 240/- (159-176). Refs: 56
ISSN: 0070-217X CODEN: CTMIA3
CY Germany
DT Journal; General Review
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
037 Drug Literature Index
LA English

L4 ANSWER 6 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 2
AN 1999:300720 BIOSIS
DN PREV199900300720
TI Modulation of an allergic immune response via the mucosal route in a murine model of inhalative type-I allergy.
AU Wiedermann, U. (1); Jahn-Schmid, B.; Repa, A.; Kraft, D.; Ebner, C.
CS (1) Institute of General and Experimental Pathology, AKH, University of Vienna, Waehringer Guertel 18-20, A-1090, Vienna Austria
SO International Archives of Allergy and Immunology, (Feb.-April, 1999) Vol. 118, No. 2-4, pp. 129-132.
ISSN: 1018-2438.
DT Article
LA English
SL English

L4 ANSWER 7 OF 64 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 3
AN 1999:181216 CAPLUS
DN 130:310306
TI Immune modulation by the cholera-like enterotoxins: from adjuvant to therapeutic
AU Williams, Neil A.; Hirst, Timothy R.; Nashar, Toufic O.
CS Dept of Pathology and Microbiology, School of Medical Sciences, University of Bristol, Bristol, BS8 1TD, UK
SO Immunol. Today (1999), 20(2), 95-101
CODEN: IMTOD8; ISSN: 0167-4919
PB Elsevier Science Ltd.
DT Journal; General Review
LA English

L4 ANSWER 8 OF 64 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 4
AN 1999:52433 CAPLUS
DN 130:250778
TI Oral ***tolerance*** and anti-pathological vaccines
AU Czerkinsky, C.; Sun, J.-B.; Holmgren, J.

CS INSERM Unit 364, Cellular and Molecular Immunology, Faculte de Medecine-Pasteur, Nice, 06107, Fr.
SO Curr. Top. Microbiol. Immunol. (1999), 236(Defense of Mucosal Surfaces: Pathogenesis, Immunity and Vaccines), 79-91
CODEN: CTMIA3; ISSN: 0070-217X
PB Springer-Verlag
DT Journal; General Review
LA English

L4 ANSWER 9 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1998:708957 CAPLUS
DN 129:335780
TI Combinations of antigen and mucosal binding component for inducing specific immunological ***tolerance***
IN Petersen, Jacob Sten
PA Zymogenetics, Inc., USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9847529	A1	19981029	WO 1998-US8361	19980423
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9871610	A1	19981113	AU 1998-71610	19980423

PRAI US 1997-44182 19970423
US 1997-44184 19970423
WO 1998-US8361 19980423

L4 ANSWER 10 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1998:124036 CAPLUS
DN 128:191574
TI Mucosal immunogens for novel vaccines
IN Russell, Michael W.; Wu, Hong-Yin; Hajishengallis, Georgios; Hollingshead, Susan K.; Michalek, Suzanne M.
PA UAB Research Foundation, USA; Russell, Michael W.; Wu, Hong-Yin; Hajishengallis, Georgios; Hollingshead, Susan K.; Michalek, Suzanne M.
SO PCT Int. Appl., 104 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9806428	A1	19980219	WO 1997-US14413	19970815
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ,				

LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
AU 9740700 A1 19980306 AU 1997-40700 19970815
PRAI US 1996-24074 19960816
WO 1997-US14413 19970815

L4 ANSWER 11 OF 64 MEDLINE DUPLICATE 5
AN 1998394389 MEDLINE
DN 98394389
TI Conditions affecting enhanced corneal allograft survival by oral immunization.
AU Ma D; Mellon J; Niederkorn J Y
CS Department of Ophthalmology, University of Texas Southwestern Medical Center, Dallas 75235, USA.
NC EY07641 (NEI)
SO INVESTIGATIVE OPHTHALMOLOGY AND VISUAL SCIENCE, (1998 Sep) 39 (10) 1835-46.
Journal code: GWI. ISSN: 0146-0404.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199811
EW 19981102

L4 ANSWER 12 OF 64 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 6
AN 1998:76750 CAPLUS
DN 128:166095
TI Cholera toxin and ***cholera*** ***toxin*** ***B*** subunit induce IgA switching through the action of TGF-.beta.1
AU Kim, Pyeung-Hyeun; Eckmann, Lars; Lee, Wha Jung; Han, Wonkyo; Kagnoff, Martin F.
CS Department of Microbiology. College of Natural Sciences, Kangwon National University, Chunchon, S. Korea
SO J. Immunol. (1998), 160(3), 1198-1203
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English

L4 ANSWER 13 OF 64 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 7
AN 1998:666307 CAPLUS
DN 130:37016
TI A plant-based ***cholera*** ***toxin*** ***B*** subunit-insulin fusion protein protects against the development of autoimmune diabetes
AU Arakawa, Takeshi; Yu, Jie; Chong, Daniel K. X.; Hough, John; Engen, Paul C.; Langridge, William H. R.
CS Center for Molecular Biology and Gene Therapy, Department of Microbiology and Molecular Genetics, School of Medicine, Loma Linda University, Loma Linda, CA, 92350, USA
SO Nat. Biotechnol. (1998), 16(10), 934-938

CODEN: NABIF9; ISSN: 1087-0156
PB Nature America
DT Journal
LA English

L4 ANSWER 14 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 8
AN 1998:184589 BIOSIS
DN PREV199800184589
TI Selective ***tolerization*** of Th1-like cells after nasal administration of a cholera toxoid-LACK conjugate.
AU McSorley, Stephen J. (1); Rask, Carola; Pichot, Roxanne; Julia, Valerie; Czerkinsky, Cecil; Glaichenhaus, Nicolas
CS (1) Dep. Microbiol., Cent. Immunol., Univ. Minn., Room 6-220, BSBE Building, 312 Church St. SE, Minneapolis, MN 55455 USA
SO European Journal of Immunology, (Feb., 1998) Vol. 28, No. 2, pp. 424-432. ISSN: 0014-2980.
DT Article
LA English

L4 ANSWER 15 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 1998:373358 SCISEARCH
GA The Genuine Article (R) Number: ZM755
TI Affinity purification of recombinant ***cholera*** ***toxin*** ***B*** subunit oligomer expressed in Bacillus brevis for potential human use as a mucosal adjuvant
AU Yasuda Y (Reprint); Matano K; Asai T; Tochikubo K
CS NAGOYA CITY UNIV, SCH MED, DEPT MICROBIOL, MIZUHO KU, NAGOYA 4678601, AICHI, JAPAN (Reprint)
CYA JAPAN
SO FEMS IMMUNOLOGY AND MEDICAL MICROBIOLOGY, (APR 1998) Vol. 20, No. 4, pp. 311-318.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0928-8244.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 16 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 9
AN 1998:170563 BIOSIS
DN PREV199800170563
TI Naltrexone effects on diazepam intoxication and pharmacokinetics in humans.
AU Swift, Robert (1); Davidson, Dean; Rosen, Seth; Fitz, Eric; Camara, Paul
CS (1) Roger Williams Med. Cent., Dep. Psychiatry, 825 Chalkstone Avenue, Providence, RI 02908 USA
SO Psychopharmacology, (Feb. 1, 1998) Vol. 135, No. 3, pp. 256-262. ISSN: 0033-3158.
DT Article
LA English

L4 ANSWER 17 OF 64 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 10
AN 1998:469280 CAPLUS

DN 129:239316
TI GM1 ganglioside-induced modulation of opioid receptor-mediated functions
AU Crain, Stanley M.; Shen, Ke-Fei
CS Department of Neuroscience, Albert Einstein College of Medicine, Bronx, NY, 10461, USA
SO Ann. N. Y. Acad. Sci. (1998), 845(Sphingolipids as Signaling Modulators in the Nervous System), 106-125
CODEN: ANYAA9; ISSN: 0077-8923
PB New York Academy of Sciences
DT Journal; General Review
LA English

L4 ANSWER 18 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 1998131447 EMBASE
TI Nasal vaccines from fundamental concepts to vaccine development.
AU Lemoine D.; Francotte M.; Preat V.
CS V. Preat, Universite Catholique de Louvain, Unite de Pharmacie Galenique, Avenue Emmanuel-Mounier 73.20, 1200 Bruxelles, Belgium
SO S.T.P. Pharma Sciences, (1998) 8/1 (5-18).
Refs: 121
ISSN: 1157-1489 CODEN: STSSE5
CY France
DT Journal; General Review
FS 026 Immunology, Serology and Transplantation
037 Drug Literature Index
039 Pharmacy
LA English
SL French

L4 ANSWER 19 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1997:735767 CAPLUS
DN 127:355338
TI ***Tolerogen*** -linked mucosa-binding molecules for immunological ***tolerance*** -inducing agents
IN Holmgren, Jan; Czerkinsky, Cecil
PA Duotol AB, Swed.
SO U.S., 13 pp. Cont.-in-part of U.S. Ser. No. 160,106, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5681571	A	19971028	US 1994-184458	19940119
SE 9303301	A	19950409	SE 1993-3301	19931008
WO 9510301	A1	19950420	WO 1994-SE941	19941007
W: AU, CA, CN, JP, KR				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2173254	AA	19950420	CA 1994-2173254	19941007
AU 9478671	A1	19950504	AU 1994-78671	19941007
AU 689138	B2	19980326		
EP 722340	A1	19960724	EP 1994-929716	19941007
EP 722340	B1	19980429		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
CN 1135182	A	19961106	CN 1994-194189	19941007

JP 09503520 T2 19970408 JP 1994-511674 19941007
AT 165515 E 19980515 AT 1994-929716 19941007
ES 2120073 T3 19981016 ES 1994-929716 19941007
PRAI SE 1993-3301 19931008
US 1993-160106 19931130
US 1994-184458 19940119
WO 1994-SE941 19941007

L4 ANSWER 20 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 11
AN 1997:255206 BIOSIS
DN PREV199799554409
TI A cholera toxoid-insulin conjugate as an oral vaccine against spontaneous autoimmune diabetes.
AU Bergerot, Isabelle; Ploix, Corinne; Petersen, Jacob; Moulin, Valerie; Rask, Carola; Fabien, Nicole; Lindblad, Marianne; Mayer, Anne; Czerkinsky, Cecil (1); Holmgren, Jan; Thivolet, Charles
CS (1) Institut National de la Sante et de la Recherche Medicale 80, Hopital Edouard Herriot, 69437 Lyon France
SO Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 9, pp. 4610-4614.
ISSN: 0027-8424.
DT Article
LA English

L4 ANSWER 21 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 12
AN 1997:345569 BIOSIS
DN PREV199799644772
TI Intranasal vaccination of humans with recombinant ***cholera*** ***toxin*** ***B*** subunit induces systemic and local antibody responses in the upper respiratory tract and the vagina.
AU Bergquist, Charlotta; Johnansson, Eva-Liz; Lagergard, Teresa; Holmgren, Jan; Rudin, Anna (1)
CS (1) Dep. Med. Microbiol. Immunol., Goteborg Univ., Gudlhedsgatan 10A, S-413 46 Goteborg Sweden
SO Infection and Immunity, (1997) Vol. 65, No. 7, pp. 2676-2684.
ISSN: 0019-9567.
DT Article
LA English

L4 ANSWER 22 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 97326097 EMBASE
DN 1997326097
TI Oral immunisation as a strategy for enhancing corneal allograft survival.
AU Ma D.; Mellon J.; Niederkorn J.Y.
CS J.Y. Niederkorn, Department of Ophthalmology, UT Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75235-9057, United States
SO British Journal of Ophthalmology, (1997) 81/9 (778-784).
Refs: 30
ISSN: 0007-1161 CODEN: BJOPAL
CY United Kingdom
DT Journal; Article
FS 012 Ophthalmology
026 Immunology, Serology and Transplantation
LA English
SL English

L4 ANSWER 23 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 97326083 EMBASE
DN 1997326083
TI Oral administration of antigen in the treatment of eye disease.
AU Williams K.A.
CS K.A. Williams, Department of Ophthalmology, Flinders University South Australia, Adelaide, SA, Australia
SO British Journal of Ophthalmology, (1997) 81/9 (714-715).
Refs: 19
ISSN: 0007-1161 CODEN: BJOPAL
CY United Kingdom
DT Journal; Editorial
FS 006 Internal Medicine
012 Ophthalmology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LA English

L4 ANSWER 24 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 1998130456 EMBASE
TI The mechanism of cholera toxin adjuvanticity.
AU Lycke N.
CS N. Lycke, Dept. Medical Microbiol./Immunology, University of Goteborg, S-413 46 Goteborg, Sweden
SO Research in Immunology, (1997) 148/8-9 (504-520).
Refs: 114
ISSN: 0923-2494 CODEN: RIMME5
CY France
DT Journal; General Review
FS 026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English

L4 ANSWER 25 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 97312749 EMBASE
DN 1997312749
TI ***Tolerance*** - Will it end in tears?.
AU Stanford M.R.
CS M.R. Stanford, The Rayne Institute, St Thomas' Hospital, London, United Kingdom
SO Eye, (1997) 11/4 (441-442).
Refs: 11
ISSN: 0950-222X CODEN: EYEEEC
CY United Kingdom
DT Journal; Editorial
FS 012 Ophthalmology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English

L4 ANSWER 26 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 13
AN 1997:180860 BIOSIS
DN PREV199799472573

TI Suppression of delayed-type hypersensitivity and IgE antibody responses to ovalbumin by intranasal administration of Escherichia coli heat-labile enterotoxin B subunit-conjugated ovalbumin.
AU Tamura, Shin-Ichi (1); Hatori, Emiko; Tsuruhara, Takashi; Aizawa, Chikara; Kurata, Takeshi
CS (1) Dep. Pathol., NIH, 1-23-1 Toyama, Shinjuku-ku, Tokyo 162 Japan
SO Vaccine, (1997) Vol. 15, No. 2, pp. 225-229.
ISSN: 0264-410X.
DT Article
LA English

L4 ANSWER 27 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1997:144284 BIOSIS
DN PREV199799443487
TI Oral ***tolerance*** in the collagen induced chondritis.
AU Kim, Nacksung; Cheng, Kuang Chaun; Lee, Kyung Mi; Yoo, Tai June
CS Univ. Tenn., Memphis, TN USA
SO Journal of Allergy and Clinical Immunology, (1997) Vol. 99, No. 1 PART 2, pp. S189.
Meeting Info.: Joint Meeting of the American Academy of Allergy, Asthma and Immunology, the American Association of Immunologists and the Clinical Immunology Society San Francisco, California, USA February 21-26, 1997
ISSN: 0091-6749.
DT Conference; Abstract
LA English

L4 ANSWER 28 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 14
AN 1998:6568 BIOSIS
DN PREV199800006568
TI Mucosal ***tolerance*** : A two-edged sword to prevent and treat autoimmune diseases.
AU Xiao, Bao-Guo; Link, Hans
CS Div. Neurol., Karolinska Inst., Huddinge Univ. Hosp., Stockholm Sweden
SO Clinical Immunology and Immunopathology, (Nov., 1997) Vol. 85, No. 2, pp. 119-128.
ISSN: 0090-1229.
DT General Review
LA English

L4 ANSWER 29 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1997:463499 CAPLUS
DN 127:99593
TI ***Cholera*** ***toxin*** ***B*** subunit-antigen conjugates for inducing immunity and/or ***tolerance***
AU Holmgren, J.; Bergqvist, C.; Czerkinsky, C.; Fredriksson, M.; Johansson, E.-L.; Lebens, M.; Lindblad, M.; Rask, C.; Sun, J.-B.
CS Goteborg University, Goteborg, S-413 46, Swed.
SO Proc. Int. Symp. Controlled Release Bioact. Mater. (1997), 24th, 113-114
CODEN: PCRMEY; ISSN: 1022-0178
PB Controlled Release Society, Inc.
DT Journal; General Review
LA English

L4 ANSWER 30 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 15
AN 1997:274536 BIOSIS

DN PREV199799566254
TI Routes of immunization and antigen delivery systems for optimal mucosal immune responses in humans.
AU Mestecky, J.; Michalek, S. M.; Moldoveanu, Z.; Russell, M. W.
CS Dep. Microbiol. Med. and Oral Biol., Univ. Alabama at Birmingham, Birmingham, AL 35294 USA
SO Behring Institute Mitteilungen, (1997) Vol. 0, No. 98, pp. 33-43.
ISSN: 0301-0457.
DT Article
LA English

L4 ANSWER 31 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1996:344999 BIOSIS
DN PREV199699067355
TI ***Cholera*** ***toxin*** ***B*** subunit as transmucosal carrier-delivery and immunomodulating system for induction of antiinfectious and antipathological immunity.
AU Czerkinsky, Cecil; Sun, Jia-Bin; Lebens, Michael; Li, Bin-Ling; Rask, Carola; Lindblad, Marianne; Holmgren, Jan
CS Dep. Med. Microbiology Immunology, Univ. Goteborg, Guldhedsgatan 10A, S-413 46 Goteborg Sweden
SO Weiner, Howard L. [Editor]; Mayer, Lloyd F. [Editor]. Annals of the New York Academy of Sciences, (1996) Vol. 778, pp. 185-193. Annals of the New York Academy of Sciences; Oral tolerance: Mechanisms and applications. Publisher: New York Academy of Sciences 2 East 63rd Street, New York, New York 10021, USA.
Meeting Info.: Conference New York, New York, USA March 30-April 2, 1995
ISSN: 0077-8923. ISBN: 0-89766-996-7 (paper), 0-89766-995-9 (cloth).
DT Book; Conference
LA English

L4 ANSWER 32 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 16
AN 1996:475441 BIOSIS
DN PREV199699204997
TI Treatment of experimental autoimmune encephalomyelitis by feeding myelin basic protein conjugated to ***cholera*** ***toxin*** ***B*** subunit.
AU Sun, Jia-Bin; Rask, Carola; Olsson, Tomas; Holmgren, Jan; Czerkinsky, Cecil
CS Dep. Med. Microbiol. Immunology, Univ. Goteborg, Guldhedsgatan 10A, S-413 46 Goteborg Sweden
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 14, pp. 7196-7201.
ISSN: 0027-8424.
DT Article
LA English

L4 ANSWER 33 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 17
AN 1996:576029 BIOSIS
DN PREV199799290710
TI Identification of an immunodominant T cell epitope on cholera toxin.
AU Cong, Yingzi; Bowdon, Hazel R.; Elson, Charles O. (1)
CS (1) Div. Gastroenterol. Hepatol., Univ. Alabama at Birmingham, 633 Zeigler Research Build., 703 S. 19th St., Birmingham, AL 35294-0007 USA
SO European Journal of Immunology, (1996) Vol. 26, No. 11, pp. 2587-2594.

ISSN: 0014-2980.
DT Article
LA English

L4 ANSWER 34 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 96:157644 SCISEARCH
GA The Genuine Article (R) Number: TV536
TI CYTOKINE EXPRESSION IN MICE ***TOLERIZED*** TO HOUSE-DUST MITE USING ***CHOLERA*** - ***TOXIN*** ***B*** -SUBUNIT
AU CLARK J (Reprint); SHEN T; SUZUKI M; CHENG K C; YOO T J
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (JAN 1996) Vol. 97, No. 1, Part 3, pp. 914.
ISSN: 0091-6749.
DT Conference; Journal
FS LIFE; CLIN
LA ENGLISH
REC No References

L4 ANSWER 35 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 96:247478 SCISEARCH
GA The Genuine Article (R) Number: UB192
TI LIMITING DISTORTION OF CATV LASERS
AU RAINAL A J (Reprint)
CS AT&T BELL LABS, 600 MT AVE, MURRAY HILL, NJ, 07974 (Reprint)
CYA USA
SO JOURNAL OF LIGHTWAVE TECHNOLOGY, (MAR 1996) Vol. 14, No. 3, pp. 474-479.
ISSN: 0733-8724.
DT Article; Journal
FS ENGI
LA ENGLISH
REC Reference Count: 16
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 36 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1996:145530 BIOSIS
DN PREV199698717665
TI Cytokine expression in mice ***tolerized*** to house dust mite using ***cholera*** ***toxin*** ***B*** subunit.
AU Clark, J.; Shen, T.; Suzuki, M.; Cheng, K. C.; Yoo, T. J.
CS Memphis, TN USA
SO Journal of Allergy and Clinical Immunology, (1996) Vol. 97, No. 1 PART 3, pp. 411.
Meeting Info.: Fifty-second Annual Meeting of the American Academy of Allergy Asthma and Immunology New Orleans, Louisiana, USA March 15-20, 1996
ISSN: 0091-6749.
DT Conference
LA English

L4 ANSWER 37 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 96:387618 SCISEARCH
GA The Genuine Article (R) Number: UJ883
TI ***CTB*** -INSULIN CONJUGATES POTENTIATE ORAL ***TOLERANCE*** AGAINST AUTOIMMUNE DIABETES IN NOD MICE
AU BERGEROT I (Reprint); MOULIN V; FABIEN N; PLOIX C; CZERKINSKY C; THIVOLET

C
SO DIABETES, (MAY 1996) Vol. 45, Supp. 2, pp. 301.
ISSN: 0012-1797.
DT Conference; Journal
FS LIFE; CLIN
LA ENGLISH
REC No References

L4 ANSWER 38 OF 64 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 18
AN 1996:304652 CAPLUS
DN 125:7628
TI ***Cholera*** ***toxin*** ***B*** subunit as transmucosal carrier-delivery and immunomodulating system for induction of antiinfectious and antipathological immunity
AU Czerkinsky, Cecil; Sun, Jia-Bin; Lebens, Michael; Li, Bin-Ling; Rask, Carola; Lindblad, Marianne; Holmgren, Jan
CS Department of Medical Microbiology and Immunology, University of Goeteborg, Goeteborg, S-413 46, Swed.
SO Ann. N. Y. Acad. Sci. (1996), 778(Oral Tolerance: Mechanisms and Applications), 185-193
CODEN: ANYAA9; ISSN: 0077-8923
DT Journal; General Review
LA English

L4 ANSWER 39 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1995:645228 CAPLUS
DN 123:31237
TI Immunological ***tolerance*** -inducing agent
IN Holmgren, Jan; Czerkinsky, Cecil
PA Swed.
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9510301	A1	19950420	WO 1994-SE941	19941007
W: AU, CA, CN, JP, KR				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
SE 9303301	A	19950409	SE 1993-3301	19931008
US 5681571	A	19971028	US 1994-184458	19940119
AU 9478671	A1	19950504	AU 1994-78671	19941007
AU 689138	B2	19980326		
EP 722340	A1	19960724	EP 1994-929716	19941007
EP 722340	B1	19980429		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09503520	T2	19970408	JP 1994-511674	19941007

PRAI SE 1993-3301 19931008
US 1993-160106 19931130
US 1994-184458 19940119
WO 1994-SE941 19941007

L4 ANSWER 40 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1996:45643 BIOSIS

DN PREV199698617778
TI Induction of immunity of mucosal surfaces: From vaccine development to specific immunotherapy.
AU Czerkinsky, Cecil (1); Holmgren, Jan
CS (1) Dep. Med. Microbiol. Immunology, Univ. Goteborg, S-413 46 Goteborg Sweden
SO Auricchio, S. [Editor]; Ferguson, A. [Editor]; Troncone, R. [Editor]. Dynamic Nutrition Research, (1995) Vol. 4, pp. 161-173. Dynamic Nutrition Research; Mucosal immunity and the gut epithelium: Interactions in health and disease.
Publisher: S. Karger AG P.O. Box, Allschwilerstrasse 10, CH-4009 Basel, Switzerland.
Meeting Info.: International Symposium Capri, Italy April 22-23, 1994
ISSN: 1021-1225. ISBN: 3-8055-6063-X.
DT Book; Conference
LA English

L4 ANSWER 41 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 95249470 EMBASE
DN 1995249470
TI Induction of IgA and IgG antibodies in vaginal fluid, serum and saliva following immunization of genital and gut associated lymphoid tissue.
AU Bergmeier L.A.; Tao L.; Gearing A.J.M.; Adams S.; Lehner T.
CS Division of Immunology, UMDS, Guy's and St. Thomas's Hospital,London SE1 9RT, United Kingdom
SO Advances in Experimental Medicine and Biology, (1995) 371/B (1567-1573). ISSN: 0065-2598 CODEN: AEMBAP
CY United States
DT Journal; Conference Article
FS 004 Microbiology
026 Immunology, Serology and Transplantation
LA English
SL English

L4 ANSWER 42 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1995:970050 CAPLUS
DN 124:84187
TI Mucosal suppression by oral pre-treatment with ovalbumin and its conversion into stimulation when ovalbumin was conjugated to cholera toxin or its B subunit
AU van der Heijden, Philip J.; Stok, Wil; Bianchi, Andre T.J.
CS Department of Immunology, Central Veterinary Institute, Lelystad, Neth.
SO Adv. Exp. Med. Biol. (1995), Volume Date 1995, 371B, 1251-5 CODEN: AEMBAP; ISSN: 0065-2598
DT Journal
LA English

L4 ANSWER 43 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
AN 95245904 EMBASE
DN 1995245904
TI Etorphine elicits unique inhibitory-agonist and excitatory-antagonist actions at opioid receptors on sensory neurons: New rationale for improved clinical analgesia and treatment of opiate addiction.
AU Crain S.M.; Shen K.-F.
CS Department of Neuroscience, Albert Einstein College of Medicine, Yeshiva

University,Bronx, NY 10461, United States
SO NIDA Research Monograph Series, (1995) -/147 (234-268).
ISSN: 1046-9516 CODEN: MIDAD4
CY United States
DT Journal; Conference Article
FS 008 Neurology and Neurosurgery
024 Anesthesiology
030 Pharmacology
037 Drug Literature Index
040 Drug Dependence, Alcohol Abuse and Alcoholism
LA English

L4 ANSWER 44 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 19
AN 1995:35269 BIOSIS
DN PREV199598049569
TI ***Cholera*** ***toxin*** ***B*** subunit: An efficient transmucosal carrier-delivery system for induction of peripheral immunological ***tolerance*** .
AU Sun, Jia-Bin; Holmgren, Jan; Czerkinsky, Cecil (1)
CS (1) Dep. Med. Microbiol. Immunol., University Goteborg, Guldhedsgatan 10A, Goteborg S-413 46 Sweden
SO Proceedings of the National Academy of Sciences of the United States of America, (1994) Vol. 91, No. 23, pp. 10795-10799.
ISSN: 0027-8424.
DT Article
LA English

L4 ANSWER 45 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 20
AN 1994:533480 BIOSIS
DN PREV199497546480
TI Effects of cholera toxin adjuvant on IgE antibody response to orally or nasally administered ovalbumin.
AU Tamura, Shin-Ichi (1); Shoji, Yuki; Hasiguchi, Kazuhiro; Aizawa, Chikara; Kurata, Takeshi
CS (1) Dep. Pathol., Natl. Inst. Health, 1-23-1 Toyama, Shinjuku-ku, Tokyo 162 Japan
SO Vaccine, (1994) Vol. 12, No. 13, pp. 1238-1240.
ISSN: 0264-410X.
DT Article
LA English

L4 ANSWER 46 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 21
AN 1994:234689 BIOSIS
DN PREV199497247689
TI Conversion of orally induced suppression of the mucosal immune response to ovalbumin into stimulation by conjugating ovalbumin to cholera toxin or its B subunit.
AU Stok, W.; Van Der Heijden, P. J.; Bianchi, A. T. J. (1)
CS (1) Dep. Immunol., Central Vet. Inst., PO Box 65, 8200 AB Lelystad Netherlands
SO Vaccine, (1994) Vol. 12, No. 6, pp. 521-526.
ISSN: 0264-410X.
DT Article
LA English

L4 ANSWER 47 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 22
AN 1994:164170 BIOSIS
DN PREV199497177170
TI Antagonists at excitatory opioid receptors on sensory neurons in culture increase potency and specificity of opiate analgesics and attenuate development of ***tolerance*** /dependence.
AU Shen, Ke-Fei; Crain, Stanley M. (1)
CS (1) Dep. Neurosci., Albert Einstein Coll. Med., 1300 Morris Park Ave., Bronx, NY 10461 USA
SO Brain Research, (1994) Vol. 636, No. 2, pp. 286-297.
ISSN: 0006-8993.
DT Article
LA English

L4 ANSWER 48 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 94:175913 SCISEARCH
GA The Genuine Article (R) Number: MZ675
TI CRYSTAL-STRUCTURE OF ***CHOLERA*** - ***TOXIN*** ***B*** -PENTAMER BOUND TO RECEPTOR G(M1) PENTASACCHARIDE
AU MERRITT E A; SARFATY S; VANDENAKKER F; LHOIR C; MARTIAL J A; HOL W G J (Reprint)
CS UNIV WASHINGTON, DEPT BIOL STRUCT SM20, SEATTLE, WA, 98195 (Reprint); UNIV WASHINGTON, DEPT BIOL STRUCT SM20, SEATTLE, WA, 98195; UNIV LIEGE, BIOL MOLEC & GENIE GENET LAB, B-4000 SART, BELGIUM
CYA USA; BELGIUM
SO PROTEIN SCIENCE, (FEB 1994) Vol. 3, No. 2, pp. 166-175.
ISSN: 0961-8368.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 42
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 49 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 93:53859 SCISEARCH
GA The Genuine Article (R) Number: KH507
TI BIODEGRADABLE MICROPARTICLES FOR ORAL IMMUNIZATION
AU OHAGAN D T (Reprint); MCGEE J P; HOLMGREN J; MOWAT A M C L; DONACHIE A M; MILLS K H G; GAISFORD W; RAHMAN D; CHALLACOMBE S J
CS UNIV NOTTINGHAM, DEPT PHARMACEUT SCI, UNIV PK, NOTTINGHAM NG7 2RD, ENGLAND (Reprint); GOTHENBURG UNIV, DEPT MED MICROBIOL & IMMUNOL, S-41346 GOTHENBURG, SWEDEN; WESTERN INFIRM & ASSOCIATED HOSP, DEPT IMMUNOL, GLASGOW G11 6NT, SCOTLAND; NATL INST BIOL STAND & CONTROLS, S MIMMS EN6 3QG, HERTS, ENGLAND; GUYS HOSP, DEPT ORAL MED & PATHOL, LONDON SE1 9RT, ENGLAND
CYA ENGLAND; SWEDEN; SCOTLAND
SO VACCINE, (1993) Vol. 11, No. 2, pp. 149-154.
ISSN: 0264-410X.
DT Article; Journal
FS LIFE; AGRI
LA ENGLISH
REC Reference Count: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 50 OF 64 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.DUPLICATE 23

AN 93186943 EMBASE
DN 1993186943
TI [Transient therapeutic arterial occlusion. Historical background and future perspectives].
DIE KONTROLLIERTE KREISLAUFSPERRE. HISTORISCHE GRUNDLAGEN UND ZUKUNFTSPERSPEKTIVEN.
AU Acevedo A.
CS Universidad de Chile, Hospital del Salvador, Av. Salvador 364,Santiago, Chile
SO Wiener Medizinische Wochenschrift, (1993) 143/7-8 (144-146).
ISSN: 0043-5341 CODEN: WMWOA4
CY Austria
DT Journal; Conference Article
FS 006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
LA German
SL English; German

L4 ANSWER 51 OF 64 MEDLINE DUPLICATE 24
AN 93114904 MEDLINE
DN 93114904
TI Comparative effectiveness of the ***cholera*** ***toxin*** ***B*** subunit and alkaline phosphatase as carriers for oral vaccines.
AU Dertzbaugh M T; Elson C O
CS Division of Gastroenterology, School of Medicine, University of Alabama, Birmingham 35294..
NC T32-AIO7051 (NIDDK)
DK28632 (NIAID)
UO1-AI28147
SO INFECTION AND IMMUNITY, (1993 Jan) 61 (1) 48-55.
Journal code: GO7. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199304

L4 ANSWER 52 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 25
AN 1993:95293 BIOSIS
DN PREV199395050489
TI Modulation of oral ***tolerance*** to ovalbumin by cholera toxin and its B subunit.
AU Pierre, Pascal (1); Denis, Olivier; Bazin, Herve; Mbella, Etondoh Mbongolo; Vaerman, Jean-Pierre
CS (1) UCL-ICP-MEXP, Avenue Hippocrate, 74, B-1200 Brussels Belgium
SO European Journal of Immunology, (1992) Vol. 22, No. 12, pp. 3179-3182.
ISSN: 0014-2980.
DT Article
LA English

L4 ANSWER 53 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 26
AN 1993:79149 BIOSIS
DN PREV199395043649

TI Chronic selective activation of excitatory opioid receptor functions in sensory neurons results in opioid "dependence" without ***tolerance***

AU Shen, Ke-Fei; Crain, Stanley M. (1)
CS (1) Dep. Neuroscience, Albert Einstein Coll. Med., Yeshiva Univ., Bronx, N.Y. 10461 USA
SO Brain Research, (1992) Vol. 597, No. 1, pp. 74-83.
ISSN: 0006-8993.
DT Article
LA English

L4 ANSWER 54 OF 64 MEDLINE
AN 93050830 MEDLINE
DN 93050830
TI Oral administration of immunomodulators and the mucosal immune system.
AU Revillard J P; Cozon G; Czerkinsky C
CS Laboratoire d'Immunologie, INSERM U80 CNRS URA 1177 UCBL, Hopital E. Herriot, Lyon, France..
SO DEVELOPMENTS IN BIOLOGICAL STANDARDIZATION, (1992) 77 31-7. Ref: 25
Journal code: E7V. ISSN: 0301-5149.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199302

L4 ANSWER 55 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 27
AN 1992:238087 BIOSIS
DN BA93:126112
TI AFTER CHRONIC OPIOID EXPOSURE SENSORY NEURONS BECOME SUPERSENSITIVE TO THE EXCITATORY EFFECTS OF OPIOID AGONISTS AND ANTAGONISTS AS OCCURS AFTER ACUTE ELEVATION OF GM1 GANGLIOSIDE.
AU CRAIN S M; SHEN K-F
CS ALBERT EINSTEIN COLL. MED., YESHIVA UNIV., BRONX, N.Y. 10461.
SO BRAIN RES, (1992) 575 (1), 13-24.
CODEN: BRREAP. ISSN: 0006-8993.
FS BA; OLD
LA English

L4 ANSWER 56 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 91:533019 SCISEARCH
GA The Genuine Article (R) Number: GF655
TI THE EARLY CELLULAR AND HUMORAL IMMUNE-RESPONSE TO PRIMARY AND BOOSTER ORAL IMMUNIZATION WITH ***CHOLERA*** ***TOXIN*** - ***B*** SUBUNIT
AU LEWIS D J M (Reprint); NOVOTNY P; DOUGAN G; GRIFFIN G E
CS ST GEORGE HOSP, DIV COMMUNICABLE DIS, LONDON SW17 0RE, ENGLAND (Reprint); WELLCOME RES LABS, BECKENHAM BR3 3BS, KENT, ENGLAND
CYA ENGLAND
SO EUROPEAN JOURNAL OF IMMUNOLOGY, (1991) Vol. 21, No. 9, pp. 2087-2094.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 28

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 57 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 28
AN 1991:227786 BIOSIS
DN BA91:119246
TI INDUCTION OF AN ENTERIC IG-RESPONSE AGAINST OVALBUMIN AND STIMULATION OF THE RESPONSE BY CHOLERA TOXIN AND ITS B-SUBUNIT IN MICE.
AU BIANCHI A T J; ZWART R J; VAN DER HEIJDEN P J
CS DEP. IMMUNOL., CENTRAL VETERINARY INST., P.O. BOX 65, 8200 AB LELYSTAD, NETHERLANDS.
SO REG IMMUNOL, (1990-1991) 3 (3), 131-138.
CODEN: REGIE3.
FS BA; OLD
LA English

L4 ANSWER 58 OF 64 SCISEARCH COPYRIGHT 1999 ISI (R)
AN 91:69975 SCISEARCH
GA The Genuine Article (R) Number: EU945
TI MANIPULATION OF INTESTINAL IMMUNE-RESPONSES AGAINST OVALBUMIN BY CHOLERA-TOXIN AND ITS B-SUBUNIT IN MICE
AU VANDERHEIJDEN P J (Reprint); BIANCHI A T J; DOL M; PALS J W; STOK W; BOKHOUT B A
CS CENT VET INST, DEPT IMMUNOL, POB 65, 8200 AB LELYSTAD, NETHERLANDS (Reprint)
CYA NETHERLANDS
SO IMMUNOLOGY, (1991) Vol. 72, No. 1, pp. 89-93.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L4 ANSWER 59 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 29
AN 1989:312901 BIOSIS
DN BA88:26631
TI THE IMMUNOLOGICAL CONSEQUENCES OF FEEDING CHOLERA TOXIN I. FEEDING CHOLERA TOXIN SUPPRESSES THE INDUCTION OF SYSTEMIC DELAYED-TYPE HYPERSENSITIVITY BUT NOT HUMORAL IMMUNITY.
AU KAY R A; FERGUSON A
CS REGIONAL IMMUNOL. SERV., ST. MARY'S HOSP., HATHERSAGE RD., MANCHESTER M13 0JH, UK.
SO IMMUNOLOGY, (1989) 66 (3), 410-415.
CODEN: IMMUAM. ISSN: 0019-2805.
FS BA; OLD
LA English

L4 ANSWER 60 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 30
AN 1988:443172 BIOSIS
DN BA86:95270
TI DOUBLE-BLIND STUDY OF THE EFFECT OF CYCLOTROPIUM BROMIDE ON ESOPHAGEAL SMOOTH MUSCLE PRESENTATION OF A TEST FOR SPASMOLYTIC EFFICACY.
AU LEPSIEN G; BUCK W
CS ABT. ALLGEMEIN- UND UNFALLCHIRURGIE, ZENTRUM CHIRURGIE, UNIV. GOETTINGEN, ROBERT-KOCH-STR. 40, 3400 GOETTINGEN.
SO ARZNEIM-FORSCH, (1988) 38 (7), 927-931.

CODEN: ARZNAD. ISSN: 0004-4172.
FS BA; OLD
LA German

L4 ANSWER 61 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS
AN 1988:18654 BIOSIS
DN BR34:7164
TI SYNTHETIC SEQUENCES OF ***CHOLERA*** ***TOXIN*** ***B*** SUBUNIT ARE IMMUNOGENIC PER OS AND DO NOT INDUCE ORAL ***TOLERANCE***.
AU PEDOUSSAUT S; GUYON-GRUAZ A; DELMAS A; HALIMI H; MILHAUD G; RIVAILLE P
CS UA 163, C.H.U. ST. ANTOINE, 27 RUE CHALIGNY, 75012 PARIS, FR.
SO THEODOROPOULOS, D. (ED.). PEPTIDES, 1986; 19TH EUROPEAN SYMPOSIUM, CHALKIDIKI, GREECE, AUGUST 31-SEPTEMBER 5, 1986. XIX+684P. WALTER DE GRUYTER: BERLIN, WEST GERMANY; NEW YORK, NEW YORK, USA. ILLUS. (1987) 0 (0), 573-576.
ISBN: 3-11-010687-6, 0-89925-310-5.
FS BR; OLD
LA English

L4 ANSWER 62 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1987:595979 CAPLUS
DN 107:195979
TI Synthetic sequences of ***cholera*** ***toxin*** ***B*** subunit are immunogenic per os and do not induce oral ***tolerance***
AU Pedoussaut, S.; Guyon-Gruaz, A.; Delmas, A.; Halimi, H.; Milhaud, G.; Rivaille, P.
CS CHU St. Antoine, Paris, 75012, Fr.
SO Pept., Proc. Eur. Pept. Symp., 19th (1987), Meeting Date 1986, 573-6. Editor(s): Theodoropoulos, Dimitrios. Publisher: de Gruyter, Berlin, Fed. Rep. Ger.
CODEN: 56ABA8
DT Conference; General Review
LA English

L4 ANSWER 63 OF 64 BIOSIS COPYRIGHT 1999 BIOSIS DUPLICATE 31
AN 1986:106855 BIOSIS
DN BA81:17271
TI GERMINATION OF SPORES FROM CLOSTRIDIUM-BOTULINUM B-APHIS AND BA-410.
AU MONTVILLE T J; JONES S B; CONWAY L K; SAPERS G M
CS EASTERN REGIONAL RES. CENT., AGRIC. RES. SERV., U.S. DEP. AGRIC., PHILADELPHIA, PA. 19118.
SO APPL ENVIRON MICROBIOL, (1985) 50 (4), 795-800.
CODEN: AEMIDF. ISSN: 0099-2240.
FS BA; OLD
LA English

L4 ANSWER 64 OF 64 CAPLUS COPYRIGHT 1999 ACS
AN 1979:477591 CAPLUS
DN 91:77591
TI Effect of stress concentrators on the strength of steel 12Kh18N10T under conditions of cooling to 4.2.degree.K
AU Novikov, N. V.; Gorodyskii, N. I.; Ul'yanenko, A. P.
CS Kiev, USSR
SO Probl. Prochn. (1979), (5), 57-9
CODEN: PPCNBG; ISSN: 0556-171X

DT Journal
LA Russian

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